With the Authors comp

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RECENT RESEARCHES

ON

ARTIFICIAL TUBERCULOSIS

IN THEIR RELATION TO THE

PATHOLOGY OF PHTHISIS.

BEING A PAPER READ AT THE LEEDS MEETING OF THE BRITISH
MEDICAL ASSOCIATION.

BY

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MDCCCLXIX.

REPRINTED FROM THE EDINBURGH MEDICAL JOURNAL FOR NOVEMBER 1869.

RECENT RESEARCHES

ON

ARTIFICIAL TUBERCULOSIS.

I PROPOSE in the following pages to give an account of some of the more important results of the experiments which have been made during the last few years as to the artificial production of tubercle, to illustrate their bearing on questions of pathology, and to endeavour to bring them into relation with the teachings of clinical experience. I have first to state as clearly as I can what are the leading facts as to artificial tuberculosis, leaving out what is unimportant, taking in all that has a bearing on my subject, whether it results from my own observations or those of others.

The literature of artificial tuberculosis is very extensive;[1] for without reckoning limited inquiries, six complete series of researches have appeared in Germany, two in England, and two in France.

Artificial tuberculosis is most easily produced in the guinea-pig, next in the rabbit; with most difficulty in the dog. The results obtained with horses and cattle are as yet doubtful.[2] For three reasons the guinea-pig is preferable; (1) because it is absolutely free from liability to natural tubercle; (2) because it is little liable to acute inflammation; and (3) because it can be inoculated with absolute certainty. Artificial tuberculosis may be produced either by the inoculation of infective material or traumatically. My experiments show, that of all inoculated materials none is more certain or more active than material taken (as vaccinators say) hot from the diseased glands of a living animal already infected. The dose required is almost infinitesimal. If a diseased gland is squeezed into a little distilled water in a capsule, and the resulting slightly turbid liquid injected into the pleura or peritoneum, you are certain of results. I have inoculated with success in a great number of situations; most frequently in the subcutaneous cellular tissue, but also underneath the conjunctiva, in the brain, and in the serous membranes. For the purpose of studying the process, the serous membranes are so infinitely preferable, that I will confine myself to the results so obtained.

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[1] My own researches as to artificial tuberculosis are contained in the Appendix to the Report of the Medical Officer of the Privy Council for the present year, and in that published in 1868. The reader will also find in those papers a detailed account of other researches on the same subject.

[2] The recent researches of M. Chauveau, not yet published in detail, show that artificial tuberculosis can be produced with the greatest facility and certainty in bovine animals.

In April 1868, Dr Wilson Fox and I published for the first time experiments showing that tuberculosis may be produced in animals traumatically. I happened to have precedence as regards the date of publication, but the experiments were made independently and at about the same time. I produced tuberculosis in the guinea-pig by the insertion of setons, Dr Fox by the insertion of various non-tuberculous products, and also by setons. This result has since been abundantly confirmed by ourselves and by others, especially by Professor Cohnheim, who produced the required injury not by setons, but by the introduction of a variety of foreign bodies, in themselves harmless, into the peritoneum. In each case a cold abscess was produced around the foreign body as the primary effect, and tuberculosis supervened.

What is the nature of the result produced, and what reason have we for identifying it with human tuberculosis? I shall consider the question at the same time with relation to the traumatic and infective form. The result of either process is to produce a definite disease—a disease which affects almost all the internal organs except the brain, and consists in the formation in them of nodules of new growth. This new growth has that peculiar structure which is common to all those diseased products which Virchow calls lymphomas, including scrofula, leucæmia, tubercle, several diseases of the lower animals, particularly the tuberculous disease of horned cattle, and farcy. Virchow calls all these lymphomas, because they all present a structure which is to be found in certain organs belonging to the lymphatic system. Virchow distinguishes lymphomas into two great classes, hyperplastic and heteroplastic. The hyperplastic lymphoma *par excellence* is scrofula, for this consists in overgrowth (resulting in degeneration) of the lymphatic glands. The heteroplastic lymphoma *par excellence* is tubercle, for it consists in the growth of nodules of material which have the same structure in parts of the body where no such structure exists. Leucæmia occupies an intermediate position, because in it we have hyperplasia of the spleen and lymphatic glands, heteroplasia of the liver, kidneys, and mucous membranes. I propose to use the word adenoid to characterize the tissue, for the following reasons:—There are in various parts of the body organs which, like the morbid growths in question, possess a structure exactly resembling, and, in fact, identical with, that of the follicles of the lymphatic glands. These organs are the Peyer's follicles, the *Trachomdrüsen* of the conjunctiva, and the Malpighian corpuscles of the spleen. In addition to these, there are other parts of the body in which the same tissue is met with. Thus, in the neighbourhood of the bronchial tubes in the lungs of the guinea-pig there are collections of adenoid tissue which lie between the bronchioles and their accompanying bloodvessels. In the pleura, immediately under the epithelium, there are gland-like, or, as I call them, adenoid bodies, consisting of collections of the tissue in question, surrounding tufts of capillary vessels, and covered with epithelium. In the peritoneum (omentum) there are similar bodies; in addition to which the vessels are surrounded by sheaths or cylinders of the same structure. Underneath the parietal peritoneum there are similar sheaths much less massive, but not less distinct in structure. In the choroid of the eye similar collections of adenoid tissue exist. Finally, during the last few months we have become familiar with the fact, that the medulla of bone consists largely of adenoid tissue, the relation of which to the bloodvessels is very remarkable. The arterioles terminate by losing themselves in the adenoid pulp, the veins commencing by wide sinuses which, like those of the spleen, are mere excavations of the pulp without distinct walls.

The reason why I have drawn attention to all these anatomical facts is this:—We have seen that all these structures agree in being formed of adenoid tissue; that tubercle (in the true sense), the new growth in leucæmia, the new growths in the tuberculous disease of cattle, have this in common—that they also consist of adenoid tissue; and that Virchow distinguishes tubercle from the rest mainly in that it is heteroplastic. I have now to say, that the tubercle produced artificially is in a certain sense hyperplastic—that is, it is an overgrowth, not a new growth. Thus, the parts most apt to be affected by tubercle are those in which the structure in question exists naturally. The tubercle nodules which are formed in the peritoneum and pleura are overgrowths of nodules which existed before; the masses of new growth in the lung are overgrowths of masses infinitely smaller which existed before; the nodules in the choroid are agglomerations of pre-existing elements, and so on. In order to see this process, you should introduce into the pleura an infinitesimal portion of water impregnated with diseased gland, as already described. In a fortnight or three weeks you have your results. Suppose you take two guinea-pigs, and inject into the pleura of one, leaving the other untouched: In the former the pleura is studded with miliary tubercles, small, but visible to the naked eye; in the latter may be studied the adenoid bodies I have described. They have the same structure as the tubercles, which are simply adenoid bodies enlarged.

This process of overgrowth of pre-existing adenoid tissue is not, however, all that we observe in artificial tuberculosis. I have already stated that nodules are found not only in the serous membranes, but in the solid organs. In the latter, the constitution of the nodules is somewhat more complicated. I will take two organs to illustrate this—the liver and the lungs. In the liver there are two very distinct forms, the leucæmic and the miliary serous. In the first the new growth assumes precisely the character of leucæmic enlargement. The organ is enormously enlarged in consequence of the growth of adenoid tissue around the bile-ducts. At the same time the epithelium of the bile-ducts grows with great activity, so that you have a combination of two things—overgrowth of adenoid tissue round the ducts, of epithelium within the ducts. In the serous form, miliary nodules of adenoid tissue grow underneath the serous membrane.[1]

In the lung there are also two forms. In tuberculized animals the lung becomes disseminated with minute nodules of lobular catarrhal pneumonia. Each nodule is extremely translucent. On making sections it is found that it consists of two materials entirely different from each other anatomically. On the one hand, the alveoli are choked with the ordinary roundish cells which are always found there;[2] while on the other the alveolar walls are thickened by the growth in them of adenoid tissue. As the disease progresses these masses of lobular pneumonia coalesce. Each mass caseates at the centre, *i.e.*, becomes opaque and soft. The disintegration goes on till a vomica is formed. It is not necessary to go further. Let me now consider what is the meaning of the process I have been describing.

What is the relation between the tuberculosis produced by inoculation and that produced traumatically? In both we have to do with infection—that is, the disease progresses, not by continuous growth, but by the distribution or dispersion of infective material from one point. This point I call the focus of infection. I assume that the communication of the disease from a part primarily affected to the rest of the body takes place in the manner indicated, *i.e.*, that it is due to the conveyance of an infective material, and therefore that we may have to do with primary and secondary lesions. Now, when you produce an injury of the external surface of such a kind as to bring about those internal changes which constitute tuberculosis, you do so by first producing local primary lesions which are of the same nature as those which result from inoculation. You produce sometimes an open sore surrounded by a base of induration; sometimes an abscess with indurated walls; sometimes no perceptible abscess, but a nodule of induration. It is therefore the induration which constitutes the primary lesion—the induration, which in both cases is the source from which the whole body is infected.

What is the nature of the infective material? On this subject various opinions are entertained by observers. Dr Waldenburg holds that the infective material consists in fine particles of the inserted substance, and that these particles being introduced into the circulation form the nuclei of miliary granulations or growths.

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[1] These nodules have been described by Villemin and others as miliary tubercles.

[2] These cell-like bodies which occupy the alveolar cavities are often called epithelial. Their relation to the epithelial lining of the alveolar walls is not known.

This may be called the capillary embolism theory. Cohnheim infers from his recent experiments that the infective material is always caseous pus. He founds this inference on the observation that in his experiments the primary lesion was always a caseous abscess.

As we have no means of seizing on the infective material, and submitting it to microscopical or chemical examination, the only way in which we can arrive at a conclusion as to its nature is that of studying the course which it follows in its distribution through the body. Let us take for the purpose a case in which tuberculization is produced by injecting a trifling quantity of diluted lymph-gland into the pleura. The course followed by the infective agent in this case may be traced with some certainty. The morbid material is introduced into the serous cavity and spreads itself over its surface. It fixes itself, however (so far as can be judged by the effects), in those parts of the serous surface where there are already masses or collections of adenoid tissue. The nodules of induration are therefore the primary lesions, the centres from which the infective agent must be distributed to the rest of the body. How does this distribution take place? Obviously it must be either by the veins or the lymphatics. I am of opinion that both are concerned, and for the following reasons:—

It has now been satisfactorily ascertained that the serous cavities are in fact lymphatic reservoirs. Both the pleura and peritoneum communicate freely with lymphatic vessels. This fact is proved, not merely anatomically, but also physiologically. Coloured liquids injected into the serous cavities during life enter the lymphatics. It is therefore scarcely possible for the lymphatic system not to be infected if the serous cavities are infected. In addition to this we have the conclusive fact, that the bronchial lymphatic glands are enlarged, and eventually undergo all those changes which characterize scrofula. I do not think, as some other observers do, that the lymphatic capillaries are specially the seat of nodules. I have already given another account of their anatomical relations; and I have made definite observations as to the behaviour of the lymphatic capillaries in those parts of the peritoneum where they can be best observed—particularly on the surface of the diaphragm. I have ascertained that the natural adenoid tissue which is there to be found underneath the serous membrane, although often existing in quantity, close to minute lymphatics, does not belong to them, but to the veins; and further, that the beautifully-defined miliary granulations which often cover the surface of the diaphragm are not in the course of lymphatics, as Klebs and Aufrecht maintain, but in the course of veins. In short, I hold that those observers have both mistaken veins for lymphatics,—a mistake which is exceedingly easy. I am therefore of opinion that the tuberculous virus is taken up by the lymphatics at every focus of infection, and carried thence to the lymphatic glands; but that it is not the channel whereby it enters the circulation.

In every case the veins offer a much readier channel for the purpose than the lymphatics. Thus, if we admit that the subserous nodules stand in the same relation to the venous radicles as the adenoid masses from which they spring, it appears certain that the facilities for the penetration of molecular morbid products or even of corpuscles must be very great; and it is further clear that, if any such material does enter the venous system, nothing can hinder its general distribution, whereas what is absorbed by the lymphatics is liable to be arrested in its passage through the lymphatic glands.

The third stage of the infective process obviously consists in the general distribution of the morbid material throughout the system by the arteries. This can be best understood by again referring to what occurs in the liver. If the injection is made into the peritoneum the surface of the liver becomes infected directly. It becomes studded with miliary subserous granulations. If the injection is into the pleura it does not at first become diseased at all, but eventually adenoid deposits are found round the ducts. The difference of form of lesion corresponds to the difference of mode of infection. Infection by the arteries gives rise to disseminated or infiltrated new growth in the depth of the organ. Infection by the peritoneum causes isolated miliary granulations on the surface.

In what way do these facts as to the course followed by the infective agent aid us in arriving at a conclusion as to its nature? I hold with Waldenburg the material to be solid matter in a state of extremely fine division, but do not agree with him in separating tuberculosis from the infective processes which constitute the specific infective diseases—*e.g.*, smallpox, scarlet fever, glanders. By proving that the infective material is necessarily solid, not in solution, we establish and strengthen the analogy with other infective diseases. It appears to me certain, from the experiments of Chauveau, that the contagium of smallpox is an insoluble substance, and exists in the state of minute particles; and that the contagia of vaccine, of glanders, and of ovine smallpox are of a similar nature. All our researches therefore tend to bring tuberculosis into the category of infective diseases; but, on the other hand, they lead us to believe that the infective matter is infinitely more common, and that the conditions for its production are probably much more of frequent occurrence than those which generate the other morbid poisons.

I have now said as much as I have time to say about the results of artificial tuberculosis. I have endeavoured to show that the disease produced is an infective disease, that its spread through the body is due to the presence in the circulation of an infective material, which may, I think, be nearly allied to the infective materials of other diseases of the same class, and that its anatomical resemblance to the so-called tuberculous diseases in man and other animals is so great that it is not possible to point out a difference.

I now proceed, by way of application, to refer to the most important of the doctrines which have been advanced of late years as to the nature of human tubercle, and to see how far the facts we have been considering bear upon them.

I need not observe that the whole of our modern notions about pulmonary tuberculosis date from Laennec. Not because he was the first that ever observed well on the subject, but because the discovery of auscultation so entirely altered the clinical aspect of the disease, that pathologists—unfortunately it must be admitted—forgot all that had preceded, or seized upon the new doctrine of tubercle as if it were definitive. There never was a more complete case of turning over a new leaf. It was not merely receiving the new, but clean forgetting the old as if it had never existed.

The new doctrine was this—That tubercle is a specific new growth, something not existing before, which grows in the affected parts; that the new growth is partly characterized by its physical properties, but mainly by the orderly succession of changes which it undergoes; that it is first gray and translucent, afterwards becomes opaque, and finally cheesy. It was Laennec who originated the distinction we now constantly hear of between gray tubercle, crude tubercle, and cheesy tuberculc. These three conditions he thought so unvarying in their relation to each other that, if in any given instance the third were found, he considered that the first and second must have preceded it.

In its application to phthisis this theory is very significant. It implies that the disease consists in consolidation, followed by disintegration of the consolidated parts. In the discussion on the nature of the disease all turns on the conclusion formed as to the nature of the first consolidation. Laennec advanced for the first time the theory that the consolidation was due to the deposit in the organ of a new material, a new kind of morbid growth. This he called tuberculous material. He said it had nothing to do with inflammation either chronic or acute, nothing to do with any other sort of consolidation except itself. How did it happen that this doctrine was so generally accepted? For reasons apparently which had no immediate relation with the question at issue. It was thought that Laennec knew more about it than any one else. He had discovered auscultation and percussion; he had taught men to make out by physical signs that phthisis existed long before the signs of marasmus were to be observed. In short, the specificity of tubercle was believed in because Laennec taught it.

It is well known that Laennec's greatest opponent was Broussais. For at that time Paris was the arena of pathological disputation, just as Edinburgh was at an earlier period. Just as Laennec excluded, so Broussais included inflammation in all his pathological ideas; but he distinguished two kinds of inflammation. He distinguished between acute inflammation, on the one hand, and those chronic indurations which result from continuous irritation on the other; and he had a theory about them, viz., that whereas acute inflammation is a disease of the blood capillaries, irritative induration is a disease of the lymphatic capillaries. In thus theorizing, Broussais went far beyond his facts, and was speculating about things beyond his knowledge, for at that time no one had seen a lymphatic capillary. To a certain extent Broussais's idea of lymphatic inflammation was a prediction or anticipation of what we now know; but after all it was a mere imagination. He saw that chronic induration was different in nature from inflammation, and therefore supposed it had a different seat. Nothing could be more natural or easy than to fix it in the lymphatic system.

Soon it appeared that Laennec's theory of specificity required some kind of support from facts. If tuberculous material were a species of morbid growth, it must have characters by which it could be recognised. These characters were sought in two directions, chemistry and anatomy. Of the efforts made by chemists some twenty years ago to characterize tubercle little need be said, for they came to nothing. The apparent results arrived at by anatomical investigation for the same purpose were much more considerable. When pathologists became possessed of the microscope one of the first uses to which they put it was to try and make out the characteristics of diseased products—so-called exudations. In Vogel's Pathological Anatomy, Gluge's Microscopical Researches in Pathology, and, above all, in Lebert's Treatise on Tubercle and Scrofula, Laennec's doctrine was, as it were, done into microscopical language. According to Lebert, tubercle consists of minute bodies of a particular size and form, from $\frac{1}{3500}$ to $\frac{1}{2500}$ inch in diameter. He made drawings of these corpuscles, and taught that by means of them the microscopist could distinguish tubercle from all other kinds of morbid product.

There can be no doubt that this great mistake of Lebert's exercised a considerable influence on pathological ideas about tubercle, and this for several reasons; most of all, because it brought the specificity doctrine of Laennec into harmony with what was then the new and prevailing pathology. Men had already made up their minds that tubercle was a specific something. Lebert gave that something an anatomical form. And it happened that the same process was going on in other departments of pathology. At the same time that the microscopists described the tubercle corpuscle they described the cancer cell and the typhoid cell—both of which were thought to be as characteristic forms of cancer and typhoid fever respectively as certain crystalline forms are characteristic of bodies of definite chemical composition.

It would probably be wrong to say that these early efforts of microscopical anatomy to elucidate pathogeny were mischievous, but they were certainly retrograde, for they were, after all, mere efforts to inspire with a new and fictitious life old and obsolete, and certainly mistaken, imaginations, by attaching them to actual anatomical forms, for Lebert and the rest saw the objects they described, and many of us had them shown to us when we were students. The success with which this was done undoubtedly gave to the doctrine of specificity a much firmer hold on the younger men of the present generation than it would otherwise have had, and even now makes it much more difficult to combat it than if we had passed at once from the antagonistic teaching of Broussais and Laennec to that of Virchow, and had been able to jump over the confusion which the microscope at first introduced into pathological ideas.

Anatomically the cardinal fact is this: All cases of phthisis pulmonalis have this in common, that they commence with lobular induration, and end in disintegration and cavern-formation. In no generally understood sense can a case of acute miliary tuberculosis be said to be a case of phthisis. This broad definition of phthisis brings us back once more to Laennec. It was he indeed who first recognised the fixed relation between induration and disintegration as antecedent and consequent; but he unfortunately extended the notion from the tissue to his "tuberculous material." The conclusion to which we have now arrived by anatomical investigation is this: There is no difference structurally between the induration of phthisis and the induration of chronic pneumonia. There are no anatomical elements in the hard parts of a phthisical lung which are not to be found in the hard parts of chronically irritated tissue in any other organ. Finally, there are no elements in the induration of phthisis which are not to be found in chronic bronchitis. It is even possible that there are no anatomical elements in the induration of phthisis which are not to be found in the healthy lung. We cannot say this positively as regards man, but it is certainly so as regards the guinea-pig.

If we examine the unsoftened part of an ordinary phthisical lung, what do we find? Precisely the same structural changes which I have described in the guinea-pig—*i.e.*, changes of two distinct kinds; those of one kind having their seat in the alveolar septa, those of the other in the alveolar cavities. The change in the alveolar septa consists in the development of the tissue which I call adenoid—lymphoid corpuscles embedded in stroma. The change in the cavities consists in accumulation of cellular elements. A diagram of the transparent nodules in the guinea-pig's lung represents as truly the early stage of tuberculous consolidation in man. In the structure itself there is nothing either specific or malignant.

In studying the development of consumption in man, it becomes more and more apparent that three distinct agencies are at work. Of these agencies one is so ill defined that we can only designate it a tendency. To some minds, I am aware, to talk of a constitutional tendency seems meaningless. I am not going to assert the possibility of disease existing absolutely independently of material change, but I do assert strenuously that disease may exist without its being possible to detect any anatomical or chemical basis for it. As regards pulmonary consumption, the fact of its so frequently attacking in succession several members of a large family shortly after adult life affords to me a proof beyond the possibility of dispute, that something phthisical exists in each such member. In this sense I believe in the existence of latent phthisis; but in no other. No proof has yet been given that, in individuals in whom this tendency exists, the solids or fluids are differently constituted from others. The latent condition can only be described as a tendency or liability, the nature of which is open to speculation.

The second agency is irritation. The organs which are subject to injury are, above all, the mucous cavities which communicate with the external air. A common bronchial catarrh, not differing in any respect except its result from other bronchial catarrhs which terminate favourably, gradually assumes the characters of consumption. In the cases in which tuberculous processes originate from catarrhal inflammations of the genito-urinary organs, the same thing happens. Gonorrhœa leads to prostatitis, prostatitis to scrofulous catarrh of the bladder, which creeps upwards along one or both ureters, and produces induration and caseation of one kidney—that condition which is called renal phthisis. In both of these instances an indurative process, which ultimately becomes disintegrative—*i.e.*, a tuberculosis—begins from a simple catarrh of irritative origin.

The third agency is that which I have designated throughout as *infection*. The word designates the fact, that whenever a chronic induration due to overcrowded corpusculation exists in any organ, it is apt to give rise to similar processes elsewhere.

Although this is the point which artificial tuberculosis serves to illustrate, the notion does not owe its origin to the results of experiment. It was distinctly formulized at least twelve years ago by Buhl almost in the terms in which we are now disposed to accept it. Buhl, however, limited its application to cases in which there are miliary tubercles. He showed, in the first place, that in persons who die of that variety of fever which is characterized by the general dissemination throughout the body of miliary granulations (acute miliary tuberculosis), masses of induration, which have remained in the body long enough to become caseous, are in the great majority of cases to be found; and *secondly*, that in those cases in which miliary tubercles are sown over a much more limited region, as, *e.g.*, when they are confined to a single organ, they also spring from old lesions.

Both of these doctrines of Buhl's are, I think, well established, and will be accepted by most pathologists, provided they are not overstated.

They do not, however, include the ordinary case of phthisis, for the instances of phthisis in which typical miliary tubercles are to be found are comparatively very few in number. The position I wish to advance is that *infection* has to do with the development of ordinary consumption—in short, that Buhl's doctrine applies in a certain sense to the so-called infiltrated forms of induration as well as to the miliary.

The results of experiment, as I have said, help us to understand this. If inoculation had produced only miliary lesions, we might have been disposed to *limit* infection to such lesions. The facts are otherwise. By inoculation you get miliary granulations in the serous membranes and in the choroid, but interstitial or diffused lesions in the liver, lungs, and other massive organs—so that here interstitial induration is part of an unquestionably infective process.

By regarding the development of so-called infiltrated gray tubercle as infective, one gets rid of some difficulties. We are no longer compelled to accept the teaching of some of the French followers of Virchow, who restrict the term tuberculosis to what is called "granulie," or to draw too marked a line of distinction between cases of phthisis in which miliary granulations are present and those in which none can be found—or, with Niemeyer, to refuse to apply the term tuberculosis to any excepting certain rare forms of consumption.

As to the word tuberculosis, obviously it would be a great advantage pathologically to get rid of it, as having no anatomical or chemical basis. At the same time, it must be admitted that it has a practical value, as implying a particular kind of malignancy. This malignancy stands in close relation to the infection of which I have been speaking, and, in particular, to that crowding together of new growth, whether catarrhal or adenoid, which is the essential character of an infective focus.

In other words, if *tuberculous* as applied to any organic disease means anything, it means destructive induration—*i.e.*, that the organ is first hardened, and then becomes softened and indurated. This, for example, is the only respect in which tuberculous disease of the testicle, of the kidney, of the bone, and of the lungs all agree.

Here I must leave off abruptly. In conclusion, let me observe, that in my judgment the question of specificity is not one of merely theoretical interest. The doctrine has, I believe, exercised an unfavourable influence both on the treatment of phthisis, and on the management of phthisical patients. It has led men to forget that consumption is influenced by the ordinary causes of inflammation, not only in its origin, but in each step of its progress; and that one most important aim in treatment must be to counteract this influence—not, of course, by returning to the antiphlogistic remedies of the past, nor indeed by any special therapeutical interference, but by bestowing on the treatment of acute catarrhal affections, whether of the pulmonary, intestinal, or genito-urinary mucous membranes, a great deal more care than has hitherto been considered necessary; and, on the other hand, by affording to the poor when actually suffering from acute disease of the lungs the means of placing themselves under protection without delay from the injurious action of cold and occupation. I hold that the principle on which hospital relief should be administered for the benefit of those who are either liable to or actually affected with phthisis is that of *prevention*, and that if hospitals for the diseases of the chest are to be of any real value, the regulations for the admission of patients ought to be so framed, that all persons affected with acute disease, whether secondary or primary, may claim immediate admission into hospital on the mere ground of urgent need, independently of the accident of a governor's recommendation.

EDINBURGH: PRINTED BY OLIVER AND BOYD.